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INTRODUCTION
Visual compromise is a common presentation of pituitary macroadenomas and is related to direct optic nerve and chiasm compression. Although the extent of visual recovery following treatment depends on the duration and severity of the visual compromise, the majority of patients experience gradual improvement in their vision. Delayed visual deterioration following treatment is typically related to either tumor recurrence or radiation-induced optic neuropathy, although visual worsening due to prolapse of the optic apparatus into a secondary empty sella has rarely been reported. In 1968, Guiot reported the first case of reversible visual deterioration associated with optic chiasm prolapse following resection of a large pituitary macroadenoma (Guiot). Based on their observations, Guiot and collaborators recommended that a "prop" be placed in the sella at the time of transsphenoidal pituitary adenoma resection to prevent progressive herniation of the optic structures. Similarly, Hardy coined the term "preventive chiasmopexy" to describe filling of the sella cavity with autologous tissue such as muscle or fat following resection of large tumors to prevent this herniation phenomenon. While optic chiasm prolapse with associated visual deterioration appears to represent a rare occurrence, its true incidence and pathophysiological basis remain uncertain. Reconstruction of the sella with autologous tissues is also widely employed as a means to prevent postoperative cerebrospinal fluid leakage with these tissues typically harvested from a secondary operative site such as the abdomen. Although not frequently reported in the pituitary literature, complications of abdominal fat graft harvest include hematoma and seroma formation as well as infection with an incidence ranging from 1-7%. At our institution, we do not routinely perform dural reconstruction following transsphenoidal resection of pituitary macroadenomas using adipose tissue to prevent cerebrospinal fluid leakage or optic chiasm prolapse. In this study, we sought to determine the incidence of optic chiasm prolapse into the sellar defect by determining the radiographic position of the optic chiasm following surgery and incidence of delayed visual deterioration.

METHODS
A retrospective review was performed for 100 consecutive patients with pituitary macroadenomas who underwent transsphenoidal resection with postoperative clinical and radiographic data greater than 6 months from the date of initial surgery (Table 1). The position of the optic chiasm was determined on sagittal MRI and defined as the distance above a line constructed between the superior aspect of the tuberculum sellae and the dorsum sellae (Figure 1). The position of the optic chiasm was compared between the preoperative MRI and the available MRI most distant from the date of surgery. Visual data was obtained from the clinical record. Dural closure was performed using a synthetic dural substitute placed as an inlay graft under the dural defect and supplemented with a thin layer of dural sealant (Tisseel®, Baxter Healthcare; Duraseal®, Covidien).
endoscopic transsphenoidal resection of pathologically demonstrated pituitary macroadenomas without packing of the tumor resection cavity using synthetic or autologous materials. Preoperative MRI demonstrated the presence of a macroadenoma with suprasellar extension in all cases with a mean tumor height dimension of 24.9mm (± 10.2) (Table 2). The average position of the optic chiasm preoperatively was 9.0mm (± 5.9) above the superior aspect of the sella turcica. The mean time between the date of surgery and postoperative MRI was 422 days (± 239). No patient reported delayed visual deterioration postoperatively and the mean position of the optic chiasm on postoperative MRI was 3.0mm (± 2.0) above the superior aspect of the sella. Despite the presence of a large intrasellar tumor resection cavity in all cases, inferior prolapse of the optic chiasm was observed on delayed postoperative MRI in only 1/100 cases and not associated with visual impairment. Preoperative evaluations revealed that 57 patients complained of visual loss prior to surgery. Tumors reported to cause visual loss were significantly larger (29.2 +/- 10.0mm) than tumors that did not inhibit vision (18.5 +/- 6.5mm, p<0.0001), and the amount they displaced the optic chiasm (11.2 +/- 6.7mm) differed significantly from the amount of displacement caused by tumors that did not affect vision (6.2 +/- 3.6mm, p<0.0001). Of the patients with initial visual loss, 48 (84.2%) reported an improvement in their vision after surgery while 7 (12.28%) reported no change in their visual status. The amount of postoperative displacement of the optic chiasm in patients whose vision did not change postoperatively was not significantly different from that of patients who experienced visual improvement after surgery. At follow-up, no change in visual status was reported for any of the patients who were without visual deficiencies before surgery. No delayed visual worsening throughout the study period was observed. No patient developed a postoperative cerebrospinal fluid (CSF) leak, though 17 experienced intraoperative leaks.

**DISCUSSION**

This study demonstrates that inferior prolapse of the optic apparatus into the sellar defect following transsphenoidal pituitary macroadenoma resection represents an extremely rare occurrence and placement of an intrasellar “prop” consisting of harvested autologous tissue is not necessary to achieve stable visual recovery. Our success, combined with the rarity of visual loss secondary to optic apparatus prolapse, lead us to reason that the risks of autograft outweigh its possible benefits.

**Pathophysiology of delayed visual loss**

Since Guiot first introduced the concept of a “prop” to prevent delayed visual changes after macroadenoma resection, several groups have tested theories regarding the pathophysiology of this complication. Chiasmal scarring was present in the fifteen surgical cases in the literature (Czech 1999, Decker 1977, Fischer 1994, Thome 2004), and is mostly accompanied by displacement of the chiasm and optic nerves into the sella. In our practice, we have seen several cases of delayed visual deterioration secondary to shrinkage of giant pituitary prolactinomas with optic chiasm prolapse following prolonged dopamine agonist therapy (Figure 2). In all cases, the visual worsening has stabilized an ultimately improved following temporary cessation of dopamine agonist therapy and slight regrowth of the prolactinoma and return of the chiasm to a more normal position, further suggesting that these invasive tumors may become tethered to the chiasm and pull the optic apparatus into the sella. Alternative proposed etiologies for delayed visual loss following pituitary tumor resection include vascular compression, scarring and radiation effects (Thome 2004) (Lee, 1983) (Adams 1988).

**Risks of autologous grafting**

Autologous grafting has long been part of closure techniques for skull base

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**Table 1: Demographics**

<table>
<thead>
<tr>
<th>Number of patients</th>
</tr>
</thead>
<tbody>
<tr>
<td>Male</td>
</tr>
<tr>
<td>Female</td>
</tr>
<tr>
<td>Average Age</td>
</tr>
<tr>
<td>Tumor type</td>
</tr>
<tr>
<td>Non-secretory Adenoma</td>
</tr>
<tr>
<td>Prolactinoma</td>
</tr>
<tr>
<td>Growth Hormone Secreting Adenoma</td>
</tr>
<tr>
<td>CSF leak</td>
</tr>
<tr>
<td>Intraoperative</td>
</tr>
<tr>
<td>Postoperative</td>
</tr>
</tbody>
</table>

**Figure 2: Optic Chiasm Position**

| Average tumor size (mm) | 24.9 ± 10.2 | 18.5 ± 6.5 | 29.2 ± 10.1 |
| Average preoperative displacement of OC (mm) | 9.0 ± 5.9 | 6.2 ± 3.6 | 11.2 ± 6.7 |
| Postoperative position of OC (mm) | 3.0 ± 2.0 | 3.1 ± 2.0 | 2.6 ± 1.7 |

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surgery, but it comes with some risks. These include an extra incision, with inherent risk of infection at donor or recipient sites, seroma formation, and donor site dehiscence. Taha et al. report on 10 complications in 974 cases in which autologous fat graft was used to reconstruct the skull base. They report this 1% rate of early and late complications secondary to fat necrosis, including sterile liquefied fat fistula, CSF leakage, and lipoid meningitis (Taha 2011). Sade adds that autologous graft can mimick tumor on imaging, making the identification of recurrence more difficult. This contrasts with surgical and fibrin glues, which produce a thin rim of hypointensity, which is distinctive from recurrence on MRI (Sade 2006). Additionally, packing the sella can cause complications secondary to mass effect, which would compromise the decompressive goal of macroadenoma resection (Slavin 1993).

CONCLUSION

These data confirm that reconstruction of the sellar defect is not necessary to prevent optic chiasm prolapse following transsphenoidal resection of a pituitary macroadenoma. Furthermore, harvest of autologous tissues such as adipose tissue is not necessary for prevention of postoperative CSF leakage, even when an intraoperative CSF leak is experienced.

REFERENCES